

Can microplastics facilitate the emergence of infectious diseases?

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1	Discussion Article
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3	Can microplastics facilitate the emergence of infectious diseases?
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Abstract

Plastic pollution is a major environmental problem. Small plastic particles (called microplastics) have been reported to have pernicious effects on human and wildlife health, by altering physiological functions (e.g., immunity, metabolism) and interfering with commensal microorganisms. However, in addition to these direct toxic effects, we suggest that microplastic pollution might also exert deleterious effects, modifying (i) the exposure to pathogens (e.g., multi-drug resistant bacteria) and (ii) the dynamics of vector-borne diseases. Therefore, we argue that microplastics should be considered as a ubiquitous environmental hazard, potentially promoting the (re)emergence of infectious diseases. The implementation of multi- and interdisciplinary research projects are crucial to properly evaluate if microplastic pollution should be added to the current list of global health threats.

1. INTRODUCTION

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Since the middle of the 19th century, plastic materials have revolutionized our daily life, for better (e.g., in medical and digital sciences), and for worse (e.g., single-use plastic items), quickly becoming a dreadful visible pollutant (Lavers and Bond, 2017). In addition to large waste, plastic pollution also involves small (<5 mm) particles (e.g., microfibers, microbeads and particles produced during the degradation of large plastic products), collectively called microplastics (Thompson et al., 2004). Microplastics are everywhere: in oceans and deep-sea (Woodall et al., 2014), in terrestrial ecosystems (Rilling and Lehmann, 2020), in the atmosphere (Fernando Amato-Lourenço et al., 2020) and in organisms ranging from zooplankton (Cole et al., 2015) to vertebrates (Carlin et al., 2020; Hou et al., 2021), including humans (Ragusa et al., 2021; Zhang et al., 2021; Fig. 1). The effect of microplastic ingestion on organismal health has been evaluated and now there is evidence showing that microplastics can indeed exert deleterious effects on several physiological and life history traits in both aquatic and terrestrial species (Blarer and Burkhardt-Holm, 2016; Cole et al., 2015; Lo and Chan, 2018; Salerno et al., 2021). A detailed and exhaustive description of the underlying mechanisms is well beyond the scope of this discussion article; however, for instance, once ingested, microplastics can

72 regulated expression of genes related to oxidative stress, (ii) inflammatory infiltration

the destruction of the structure of the intestinal mucosa (see Huang et al., 2021 for a

and production of pro-inflammatory cytokines such as TNF-α, IFN-γ and IL-6, or (iii)

induce the mechanical disruption of the intestinal barrier integrity, causing (i) an up-

recent review). Other direct toxic effects arise from the release of additives, plasticizers,

trace elements and other chemicals that can provoke endocrine and inflammatory disorders once microplastics are ingested (see recent reviews: Bradney et al., 2019; Callaghan et al., 2021; Jiang et al., 2020; Yilmaz et al., 2020). That said, a proper assessment of the potential deleterious effects of microplastic ingestion requires taking into account environmentally-relevant concentrations of plastic particles (i.e., those to which organisms are likely exposed in their natural environment during their lifespan).

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Another important methodological issue when it comes to the assessment of the health consequences of microplastic pollution is related to the use of virgin microplastics versus aged particles, the reason being that microplastics released in the environment are readily colonized by a diversity of microorganisms. More specifically, several studies have reported that microplastics can carry different bacterial communities depending on their types (Steinman et al., 2020) and have also different composition and properties (antibiotic resistant bacteria and antibiotic resistance genes) compared to bacteria found in the surrounding environment (Oberbeckmann et al., 2018; Wang et al., 2021; Wu et al., 2019a; Zhang et al., 2019). Due to their persistence in the environment, microplastics might therefore play a role in spreading potentially harmful bacteria (both for humans and animals) across different environments (marine, freshwater, terrestrial). This would represent an additional, indirect, impact of microplastics on organismal health (Bowley et al., 2021; Prata et al., 2021). In addition, there are several reasons to think that microplastics might also alter the capacity of arthropod vectors to transmit a diversity of pathogens, through their effects on vector microbiota and immunity, or through the effects on interacting species (predators, competitors).

Despite the concern over the emergence and reemergence of infectious diseases at the local and global scales (Morens and Fauci, 2013; Plowright et al., 2017), the possibility that microplastic pollution might facilitate the spread of pathogenic microorganisms and alter the dynamics of vector-borne diseases have been barely discussed and investigated. In this discussion article, we would like to highlight the link between two current global threats that are the increase in microplastic pollution and the acceleration of disease emergence. There are numerous unknown indirect effects of microplastics that need to be investigated to evaluate if and how microplastic pollution might promote the emergence of novel pathogens, potentially affecting human, livestock and wildlife health. Here, we indicate some research directions that might prove useful to assess the risk that microplastic pollution might exert as promoter of infectious disease (re)emergence. We focus on microplastics because of their ubiquitousness in the environment and their small size, which allows them to be ingested by a large diversity of aquatic and terrestrial organisms. We think they have the potential (i) to act as disease vehicles into organisms more easily than large plastic waste and (ii) to provoke physiological disorders into vectors of infectious diseases.

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2. MICROPLASTICS CAN ACT AS VECTORS OF ANTIBIOTIC

RESISTANT BACTERIA

- 121 2.1. Increased exposure to antibiotic resistant bacteria
- 122 Virtually all human beings are today continuously exposed to plastic debris through
- ingestion and inhalation (Zarus et al., 2021). A large amount of evidence has rapidly
- accumulated showing that we ingest microplastics on a daily basis: several food items
- and drinking water have been shown to be contaminated by microplastics (Zhang et al.,

2021). Similarly, plastic debris with a size < 1µm are suspended in the air and can be inhaled (Fernando Amato-Lourenço et al., 2020; Prata, 2018). Estimates indicate that people living in highly urbanized areas inhale microplastic particles daily (Wright et al., 2020) and this also applies to domestic indoor environments (Vianello et al., 2019). The inhalation route has been corroborated by the finding of plastic particle contamination in human lung biopsies (Fernando Amato-Lourenço et al., 2021).

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Knowing all of this, an important parameter to take into consideration is that living beings get exposed through ingestion and inhalation not only to the microplastics but also to the microorganisms that live on them. The term "plastisphere" was coined by Zettler and colleagues to describe the community of bacterial species that was found on marine plastic debris in the North Atlantic subtropical gyre (Zettler et al., 2013). The main result of their work, corroborated by several other studies, was that microorganisms living on microplastics were not a subsample of the surrounding environment, but rather showed a distinct diversity, composition and functional properties (Amaral-Zettler et al., 2020; Mammo et al., 2020; Wang et al., 2020; Wu et al., 2019a; Xue et al., 2020). Microplastics have several features that promote bacterial colonization compared to the surrounding environment and natural substrates (Amaral-Zettler et al., 2020; Wu et al., 2019a). In particular, bacteria can readily form a biofilm on microplastics, which provides shelter and increases bacterial survival in the face of environmental stressors (Laverty et al., 2020; Wang et al., 2020). Antimicrobial resistance is one of the major health challenges that we will face in the upcoming years (Cassini et al., 2019; Collignon et al., 2018). Several studies revealed that microplastics selectively enrich antibiotic resistant bacteria and genes (ARBs and ARGs; Liu et al., 2021; Sathicq et al., 2021; Wang et al., 2020). Evidence shows that microplastics can harbor microbial communities carrying ARGs with abundances 100 to 5,000 times higher than planktonic bacteria (Zhang et al., 2020), although the process of ARGs enrichment depends on the features of the microplastics and their ageing (Su et al., 2021). Since biofilm formation favors horizontal gene transfer, microplastics can become hotspots for ARGs because resistance genes carried by mobile elements can spread more easily in the biofilm compared to planktonic bacterial communities (Arias-Andres et al., 2018). Microplastics can also have affinity for antibiotics that are released in the environment (Li et al., 2018) and for other chemical pollutants (Scott et al., 2021). For example, antibiotics and trace metals can attach to microplastics and exert selection pressures on bacteria living on plastic debris, and may also have a synergistic effect on bacterial resistance due to co-selection (Imran et al., 2019; Yang et al., 2019; Fig. 2). Such synergistic effects can result in strengthened selection on resistant strains, and improve the resistome of microplastic-associated bacteria. Although most studies have focused on microplastics in marine environments, the vast majority of plastic particles come from land-based human activities (Xu et al., 2020). Therefore, the distribution of microplastics can be seen as a mosaic of pollution hotspots from which particles flow into other environments. Several hotspots of microplastic pollution have been identified such as urban wastewater treatment plants (WWTP) (Syranidou and Kalogerakis, 2022). These hotspots of microplastic pollution are however, also hotspots for antibiotics, trace elements, ARBs and ARGs (Rizzo et al., 2013; Zhang et al., 2022). In these environments, microplastics might then serve as sinks for other pollutants and ARBs, and spread them in other environments via leachate, landfill leakage, effluents of WWTPs, sewage sludge (Kelly et al., 2021). Current estimates indicate that 25% of microplastics found in marine environments come from WWTPs (Syranidou and Kalogerakis, 2022). However, although

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microplastics can successfully contribute to spread ARBs (homogenizing the risk of disease emergence), other environmental factors do contribute to the mosaic of disease emergence risk, including proximity to pollution hotspots and human population density. With this respect, urban WWTPs and landfills should be considered as particularly risky sites, especially when in proximity of megalopolis. Urban environments, highways and roads also produce large amounts of microplastics, such as tire particles that are subsequently found in the soil (Sommer et al., 2018). As other microplastic substrates, tire particles have been shown to harbor microbial communities that differ from the surrounding soil. Bacteria colonizing tire particles also have different sets of ARGs and, of more concern, the abundance of ARGs is enhanced when tire particles are exposed to soils containing other pollutants such as heavy metals and antibiotics (Ding et al., 2021).

2.2. Risks associated with increased exposome and resistome

Microplastics have been reported to carry pathogens of aquatic organisms (fish, crustaceans, mollusks). Isolates of the fish pathogen *Aeromonas salmonicida* expressing virulence factors related to the secretion systems Type II and IV, and several toxins have been retrieved from plastic debris (Radisic et al., 2020; Viršek et al., 2017). These isolates also carried antimicrobial resistance genes (lactamases and chloramphenicol acetyltransferase), which raise the question of the potential impact that microplastics might have as disease vectors in aquaculture (Dong et al., 2021). In the same line, an incubation experiment showed that, compared to surrounding seawater, mariculture-derived microplastics harbor (i) distinct bacterial communities as the exposure time increases, (ii) a higher abundance of bacteria of the genera *Vibrio*, *Pseudoalteromonas* and *Alteromonas* (Sun et al., 2020). The idea that microplastics

might be vehicles of pathogenic microorganisms in aquaculture systems is further supported by recent evidence showing that oysters (Oestra edulis) have a higher uptake rate of microplastics coated with bacteria compared to virgin particles. In addition, oysters exposed to bacteria-coated microplastics have increased oxygen consumption and respiration rate compared to oysters exposed to virgin microplastics (Fabra et al., 2021). Therefore, benthic filter feeders are likely to be preferentially exposed to bacteria-carrying microplastics and as such transfer them over the trophic chain, up to humans. Similarly, several human pathogens (including opportunistic ones) have been discovered on plastic debris in different geographical regions and environments (Table 1). For instance, enteropathogenic strains of Vibrio and Escherichia coli have been reported on microplastics sampled at the Guanabara Bay (Brazil) while water samples had low bacterial titers (Silva et al., 2019). Hence, microplastics can substantially contribute to spread such pathogens from areas with fecal bacteria contamination to preserved zones. If these preserved zones involve recreational environments, such as beaches and bathing waters, we might easily predict an enhanced human exposure to pathogenic organisms conveyed by microplastics (Keswani et al., 2016). We suggest that the combined effect of microplastic-associated exposome and resistome might enhance the risk of emergence of novel bacterial pathogens. Although increased exposure and increased antimicrobial resistance alone represent risk factors for the emergence of bacterial diseases, microplastics might play the role of catalyzers favoring the exposure to resistant strains. In addition to this, microplastics carrying ARBs and ARGs might contribute to spread resistance genes over large distances, even in areas with no antibiotic pollution and to homogenize globally the risk of emergence of bacterial pathogens (Fig. 2). The example of polystyrene recovered in Antarctica,

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harboring microorganisms able to resist multiple drugs, including cephalosporin and quinolone (Laganà et al., 2019) should act as a serious red flag.

Although most of the evidence supporting the role of microplastics as vectors of pathogens comes from aquatic environments, the possibility that particles suspended in the air might also serve as vectors for pathogens, including airborne viruses, cannot be discarded. For instance, it has been suggested that SARS-CoV-2 might have longer survival on air suspended plastic particles than in the aerosol, contributing to the transmission and the spread of the pathogen (Liu and Schauer, 2021). ARGs have already been reported in airborne bacteria sampled in a WWTP in China, including the opportunistic human pathogens *Comamonas* and *Moraxella* (Li et al., 2016). If these airborne bacteria can use microplastics as aquatic microorganisms do, there might be a risk of environmental contamination well beyond the surroundings of WWTPs.

2.3. Summary

Here, we argued that microplastic pollution is likely to pose a threat to human and wildlife health, not only because of direct toxic effects due to the ingestion of plastic particles, but also due to the exposure to pathogenic microorganisms carrying antimicrobial resistance genes. Our argument is based on a series of well-established observations showing that microplastics harbor communities of microorganisms that 1) differ from those found on natural substrates or as planktonic forms, and 2) present a greater abundance of ARBs and ARGs. From these two observations, we consider that microplastics, by increasing the contact rate between hosts and pathogens, could facilitate the emergence of multi-drug resistant bacterial pathogens and pose a threat to humans, livestock and wildlife species. Actually, we might draw a parallel with the recent emergence of bacterial pathogens associated with the use of medical devices

(such as prostheses and catheters). Bacteria can form biofilms on such devices (Pelling et al., 2019; Stickler, 2008; Tande and Patel, 2014) as they do on microplastics and several new pathogens are now associated with the use of urinary catheters (*Aerococcus* spp., *Actinobaculum schaalii*, *Alloscardovia omnicolens*; Vouga and Greub, 2016). We suggest that the potential health and economic costs associated with the emergence of novel multi-drug resistant microorganisms is worth to stimulate more research in this area, as to better quantify the risk associated with microplastic pollution.

3. MICROPLASTICS CAN ALTER VECTOR-BORNE INFECTIOUS

DISEASE TRANSMISSION

Emerging pathogens have been associated not only with antimicrobial resistance, but also with the spread of arthropod vectors (Kilpatrick and Randolph, 2012). In fact, in addition to parasites that are directly or environmentally transmitted, wildlife and humans are exposed to multiple vector-borne pathogens, many of them being considered as emerging or reemerging (e.g., Dengue, Zika, West Nile viruses; Jones et al., 2008; Morens et al., 2004). Historically, vector control has relied on synthetic insecticides and environmental management of breeding sites, but several novel vector control tools are under development, such as the gene-drive technology or bacterial infection of vectors (e.g., *Wolbachia*; Wilson et al., 2020). We suggest that microplastic pollution, as a ubiquitous environmental hazard, should be taken into account in vector control strategies since it could interfere either with life history traits of vectors and/or their microbiota.

3.1 Alteration of vector life history traits

Many medically important insect-vectors or intermediate hosts of infectious diseases (i.e., mosquitoes, black flies, snails) are freshwater organisms inhabiting lakes, rivers, swamps or coastal ponds that can be heavily polluted with microplastics (Akindele et al., 2020; Wagner and Lambert, 2018; Windsor et al., 2019;). Microplastics contamination is not uniformly distributed around the globe and varies with freshwater ecosystems. Although lakes are generally more contaminated than rivers, and riverine and lacustrine sediments are more contaminated than the aquatic matrix (Cera et al., 2020), microplastics in lotic ecosystems are more studied than in lentic systems. In addition, invertebrates are largely understudied compared to vertebrates (i.e., fish; Cera et al., 2021). Nonetheless, it has been shown that freshwater invertebrates have the capacity to ingest microplastics, the quantity of uptake depending on their feeding type and morphology (Scherer et al., 2017; Setälä et al., 2016). Interestingly, experimental work found that adult mosquitoes do not avoid water contaminated with microplastics when ovipositing, and that larvae do ingest microplastics that remain at the adult stage with, however, no impact on their survival rate (Al-Jaibachi et al., 2018; Al-Jaibachi et al., 2019; Cuthbert et al., 2019). Depending on the vector species and their ecological niches, microplastics might have variable influence on vector's life history traits and population dynamics. Even so, we can expect various outcomes of the microplastic ingestion by the vectors, either direct effects on development, growth or survival, or indirect effects on vectorial capacity (Fig. 3). Vectorial capacity (VC) can be defined as a measurement of the efficiency of vector-borne disease transmission (Garrett-Jones et al., 1964), corresponding to the basic reproductive rate of a vector-borne pathogen. Vectorial capacity can be assessed using the following equation:

$$VC = \frac{ma^2bp^N}{-\ln(p)}$$

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where m refers to vector density, a to vector biting rate, b to vector competence (the susceptibility of the vector to become infected and transmit the pathogen), p to the probability of vector daily survival, and N to the pathogen extrinsic incubation period (Cansado-Utrilla et al., 2021). Therefore, when vectors get easily infected, have high survival rates, high density, and bite at high rate, they effectively transmit the disease. We propose that microplastics have the potential to affect all these parameters (e.g., impairing vector survival, or decreasing extrinsic incubation rate).

The effect of microplastic pollution on vector-borne diseases can be nonetheless particularly difficult to predict because of the complex ecological interactions that operate at different temporal and spatial scales. Microplastics might decrease parasite transmission if they have negative effects on vectors (reducing fecundity and/or survival). It has been well documented, for example, that decreasing the survival of adult *Anopheles* (the mosquitoes that transmit human malaria) reduced their vectorial capacity (Brady et al., 2016). Negative impacts of microplastics have also been reported on food intake, growth, reproduction or survival in freshwater invertebrates (Foley et al., 2018). Therefore, snail-borne parasitic diseases, such as fascioliasis or schistosomiasis, could be less prevalent if microplastic pollution increases aquatic snail mortality and/or decreases cercariae release in water.

3.2. Modification of vector microbiota

Microplastics, and the associated 'plastisphere', might interact with the vector microbiota and immune system and consequently increase the vectorial capacity. Recent work has focused on the effects of microplastics on the microbiota (Fackelmann et al., 2019) and the immune system in model organisms - in mice (Jin et al., 2019; Lu

et al., 2018) and zebra fish (Jin et al., 2018; Limonta et al., 2019; Qiao et al., 2019) and invertebrates (Ju et al., 2019; Zhu et al., 2018). For example, it has been shown that ingestion of microplastics by dipteran larvae triggers the activation of phenoloxidase, one of the effectors of innate immunity (Silva et al., 2021a), but also increases oxidative damage (Silva et al., 2021b). Therefore, we might expect that insect vectors (such as black flies transmitting nematodes causing river blindness, or mosquitoes transmitting Plasmodium causing malaria) could have an altered vectorial capacity if the ingestion of microplastics affects their physiology (e.g., the immune response) and/or the microbiota. In fact, the microbiota has an important role in modulating the mosquito immune response and can alter susceptibility to infection through either immune priming or direct inhibition (Caragata et al., 2019). It has been shown that the removal of the midgut bacteria through antibiotic treatment led to a greater susceptibility of Anopheles gambiae to Plasmodium falciparum and of Aedes aegypti to dengue virus infection (Dong et al., 2009; Xi et al., 2008). Bacteria can also influence mosquito competence by impairing pathogen infection (Cirimotich et al., 2011a; Cirimotich et al., 2011b). On the contrary, the presence of members of the Enterobacteriaceae family in the mosquito midgut favors *Plasmodium* infection (Boissière et al., 2012), while another recent study reported that Aedes mosquitoes harboring a Serratia marcescens strain were more permissive to dengue virus infection than those free of S. marcescens (Wu et al., 2019b). These few examples show the complexity of the interactions between pathogens and microbiota that could affect mosquito vectorial capacity. Considering that microplastics often lead to dysbiosis and alter immune responses, we suggest that mosquitoes, or other arthropod vectors, ingesting microplastics could see their susceptibility to pathogens decrease or increase, depending on the bacteria and contaminants that microplastics carry (Fig. 3). Finally,

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little is known regarding how the microbiome influences the extrinsic incubation period (EIP; Cansado-Utrilla et al., 2021), but it has been shown that *Wolbachia* could increase the EIP of the dengue virus in *Aedes aegypti* (Carrington et al., 2018). It cannot be excluded that on the opposite, the plastisphere could shorten the EIP and have a positive effect on pathogen transmission.

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3.3. Changes in the trophic chain

We propose that microplastics might have even more subtle effects on the transmission of vector-borne diseases, by altering the population dynamics of interacting species (predator - prey, competing species). Even if microplastic pollution has no direct effect on vector species, it may have one on natural enemies that play an essential role to regulate the abundance of vectors, which is one of the factors, that influences the vectorial capacity. Pathogen transmission could drastically increase if natural enemies fail to regulate the vector proliferation (Fig. 3). For example, numerous aquatic insects or amphibians are regulating mosquito larvae density either by direct predation (Quiroz-Martínez and Rodríguez-Castro, 2007) or even by indirect stress caused by predators (Bellamy and Alto, 2018). A study found that mosquito size, fecundity and longevity decreased with indirect effects of predation, which would result in a significant decrease of malaria transmission (Roux et al., 2015). On the opposite, if predators are negatively affected by microplastic pollution, vectors would benefit from it and see their vectorial capacity enhanced. In agreement with this hypothesis, a recent meta-analysis showed that feeding activity of fish, such as the predatory performance or number of ingested prey, was consistently impaired in the presence of microplastic pollution (Salerno et al., 2021). Finally, human-driven alteration of top-down regulation of vector populations has also been reported. An emblematic example is the building of a dam on the Senegal River that was associated with an outbreak of schistosomiasis, due to the interference with the migration of prawns, one of the main predators of the snails that are the intermediate hosts of schistosome parasites. Reintroduction of prawns resulted in reduced abundance of infected snails and reduced prevalence of schistosomiasis (Sokolow et al., 2015).

Despite the manifold reasons to expect an effect of microplastic pollution on the dynamics of infectious diseases, studies on the relationship between the exposure of microplastics and risk of parasite infection in wildlife, livestock or humans are so to speak inexistent. Only two correlative studies found positive association between accumulation of microplastics and presence of parasites - in grey seals (Hernandez-Milian et al., 2019) and fishes (Grazia Pennino et al., 2020), and only one experimental study investigated the effects of microplastic ingestion in tadpoles and trematodes (Buss et al., 2021). The latter study performed an experimental microplastic exposure during one day (i.e., 24h) and found that individuals that ingested microplastics did not show a significant increase in parasite infection risks. However, one can argue that the time of exposure might not reflect the likely experience of wild organisms, which are rather chronically exposed to this pollutant.

3.4. Summary

Here, we argued that microplastic pollution is likely to modify vector-borne disease dynamics via two pathways, either by altering component of the vectorial capacity or by modifying the interactions between species along the trophic chain.

First, our argument is based on studies showing that arthropods, either as larvae or adults, are capable of ingesting microplastics, which are known to interfere with the

microbiota of organisms. Because infection dynamics of pathogens (viruses or parasites) are regulated by the microbiota, we hypothesize that microplastics could facilitate the emergence of vector-borne diseases by increasing the vectorial capacity. Second, we believe that even if microplastics do not have a significant effect on the vector microbiota and immunity, they might have undesirable consequences on predators and/or competitors, which could fail to regulate vector populations.

To date, and to our knowledge, no study has investigated the impact of microplastics on vectorial capacity, which is surprising given the possible health and economic consequences. More research in this area should be performed to properly evaluate the emergence risk of vector-borne diseases associated with microplastic pollution.

4. RESEARCH DIRECTIONS

We argued here that microplastic pollution might promote infectious disease emergence through different pathways. Although there exists evidence supporting this view, we realize that the link between microplastics and the dynamics of infectious diseases (in particular vector-borne diseases) has been barely investigated. As such, we hope that this work will contribute to stimulate further investigation to better understand how microplastics may interfere with antimicrobial resistance and the (re)emergence of vector-borne diseases.

Whatever the aspect of microplastic contamination we want to address, it is worth reminding ourselves that whether in laboratory conditions or in the field, environmentally realistic concentration of microplastics should be used (Lenz et al., 2016), while avoiding primary and pristine microplastics that are not representative of the contamination *in situ*. The age, shape, size and composition (additives and

contaminants) of microplastics as well as their bacterial communities are parameters that should be better apprehended (Waldman and Rillig, 2020). We realize however that identifying environmentally-relevant microplastic concentrations is already a challenging task because it does not only depend on the presence of microplastics in the environment but also on the actual exposure of organisms (spatially and temporally), which is linked to ecological and physiological traits of the studied species (e.g., feeding habits, lifespan). This suggests that environmentally-relevant concentrations might even differ between species living in the same areas. Our knowledge on the many-fold impacts of microplastic pollution on virtually all levels of biological organizations (from gene expression to ecosystem functioning) is rapidly expanding. Research work has been built on a variety of species, environments, experimental contexts, and has provided indisputable evidence showing that microplastics do represent a major environmental concern. However, as we learn more, we think it is time to place greater emphasis on the possible role that microplastic pollution might also play as promoter of infectious diseases emergence. Taking this into account, we would like to provide some suggestions for future research. How does urbanization influence bacteria colonization on microplastics and infection

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444 risk?

> We need to better understand how the process of microplastic colonization by microorganisms changes along the gradient of anthropogenic disturbance. Heavily polluted environments are hotspots for multi-drug resistant pathogens, however microplastics have the potential to spread them at very large geographical scales. Long term surveys of bacteria richness and diversity on microplastics should be performed in soil and aquatic environment along gradient from cities and intensive agricultural

lands where the trace metals and antibiotics are most likely to occur, to wilderness areas that present low human population density. This might allow us to draw risk maps, providing a useful tool to guide decisions on where monitoring effort should be prioritized.

Can microplastics biofilm interfere and undermine mosquito control methods?

Future work should focus on the possible effects of microplastics on vector control strategies. *Wolbachia*-based strategy for controlling the transmission of dengue and other arboviral infections has been largely investigated and field releases have already occurred in different places (Kamtchum-Tatuene et al., 2017). However, microplastics might be able to modify bacterial communities and/or their interactions, and potentially wipe off the suppressive effects of *Wolbachia* on arboviroses. Biocontrol strategies should definitely take into consideration this increasing environmental pollution.

Does aquatic environment influence interaction between microplastics and population

dynamics of vectors?

More effort should be put in the understanding of the environmental traits that might act in synergy with microplastic pollution. For instance, microplastics present in freshwater versus brackish water, or in small versus large body of water might affect differently the population dynamics of vectors. This is not a trivial point since many species of insect vectors or intermediate hosts have different ecological requirements and habitat preferences. We should investigate which ecological and biogeographical parameters are more likely to impact vector and intermediate host life history traits and vectorial capacity in the context of chronic exposure to microplastics.

abundance and/or behavior of natural enemies?

We advocate for a more holistic approach to the study of microplastics, that explores the effects at the ecosystem level. Up to now, most of the studies have focused on the direct effect of microplastic pollution on the species of interest. Nevertheless, the abundance of vectors and intermediate hosts of human infectious diseases can dramatically change if predators decline in number in polluted areas. We suggest that future work should implement a more integrative approach to assess the effect of

Do microplastics change the population dynamics of vectors by reducing the

5. CONCLUSIONS

microplastics on ecosystem functioning.

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Microplastics are clearly not a classical pollutant, in the sense that they are ubiquitous and highly persistent, which make them today an accepted indicator of the anthropocene (Zalasiewicz et al., 2016). Evidence is rapidly accumulating showing that microplastics can have pernicious health effects on organisms that ingest/inhale them due to their direct toxicity. In this discussion piece, we draw the attention to an additional pernicious effect of microplastic pollution. We argue that microplastics can facilitate the emergence of infectious disease by increasing the exposure of bacterial pathogens to human, livestock and wildlife and by altering the dynamics of vector-borne infections. We suggest that if microplastics do play a role promoting the (re)emergence of infectious diseases (both multi-drug resistant bacteria and vector-borne pathogens),

they will pose a more serious threat in terms of public health than contaminants absorbed by microplastics (e.g., persistent organic pollutants; Amelia et al., 2021). Our argument is based on two lines of reasoning: first, once an infectious disease has emerged, the whole population of susceptible individuals is potentially at risk, irrespective of whether they are exposed to the source of the emergence or not. The COVID-19 pandemic perfectly illustrates this point. Although SARS-CoV-2 is supposed to have initially infected humans exposed to animals carrying the virus, at a very local scale, human-to-human transmission rapidly made the whole human population at risk (including those not exposed to SARS-CoV-2 reservoirs). Obviously, this argument does not apply to non-transmissible diseases induced by contaminants. The second line of reasoning relies on the potential that microorganisms have to adapt to novel environmental conditions through evolutionary changes. These evolutionary changes might contribute to increase (or decrease) infectivity and virulence, depending on the selective forces operating on the pathogens and the epidemiological context. Again this provides a very different scenario compared to inert toxic compounds that are not expected to become more toxic in response to natural selection.

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We are convinced that interdisciplinary research projects are necessary to address all these questions and to propose global maps of emergence risks of infectious diseases associated with microplastic pollution, as it already exists with climate change. We are aware that such investigations are difficult to launch because it has to bring together experts from different biological fields, such as ecotoxicologists, microbiologists, ecologists, chemists, epidemiologists and modeling scientists. Because the framework that allow such holistic approach already exists into the 'One Health' concept (Zinsstag

525	et al., 2011), researchers should be encouraged to develop studies both in controlled
526	environments and in situ in order to anticipate future public health issues.
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535	C.L. and G.S. conceived and wrote the manuscript together. C.L. produced the paintings
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537	
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Table 1. A non-exhaustive list of bacterial species that can potentially cause human diseases (including opportunistic pathogens) found on microplastics. We report the location and the environmental type to illustrate the diversity of sites and environments where microplastics have the potential to carry pathogenic bacteria.

Class	Species	Location	Environmental type*	Reference
Alpha Proteobacteria	Brevundimonas spp	China (Beijing)	sewage	(Zhao et al., 2020)
Beta Proteobacteria	Aquabacterium spp	China (Beijing)	sewage	(Zhao et al., 2020)
	Neisseria spp	China (Beijing)	sewage	(Wang et al., 2021)
Gamma Proteobacteria	Acinetobacter beijerinckii	Norway (Øygarden)	intertidal zone	(Radisic et al., 2020)
	Acinetobacter spp	China (Guangzhou)	lake water	(Gong et al., 2019)
	Acinetobacter spp	China (Urumqi river)	river water	(Xue et al., 2020)
	Aeromonas popoffii	Norway (Øygarden)	intertidal zone	(Radisic et al. 2020)
	Alteromonas spp	China (Sungo Bay)	mariculture, shoreline	(Sun et al., 2020)
	Enterobacter spp	Ireland (Carlow)	river	(Murphy et al., 2020)
	Escherichia coli	UK (Scotland)	bathing beaches	(Rodrigues et al., 2019)
	Escherichia coli	Brazil (Guanabara Bay)	mangrove, agricultural, urban areas	(Silva et al., 2019)
	Escherichia coli	Ireland (Carlow)	river	(Murphy et al., 2020)
	Legionella spp	China (Beijing)	sewage	(Zhao et al., 2020)
	Legionella spp	China (Beijing)	sewage	(Wang et al., 2021)
	Morganella morganii	Norway (Øygarden)	intertidal zone	(Radisic et al., 2020)
	Pseudoalteromonas spp	China (Haihe estuary)	estuarine	(Li et al., 2019)
	Pseudoalteromonas spp	China (Sungo Bay)	mariculture, shoreline	(Sun et al., 2020)
	Pseudomonas aeruginosa	Texas (Gulf of Mexico)	coastal	(Sun et al., 2021)
	Pseudomonas mendocina	China (Haihe river)	river water	(Wu et al., 2019a)
	Pseudomonas monteilii	China (Haihe river)	river water	(Wu et al., 2019a)
	Pseudomonas spp	Singapore	coastline	(Curren et al., 2019)
	Pseudomonas spp	China (Urumqi river)	river water	(Xue et al., 2020)
	Vibrio cholerae	Brazil (Guanabara Bay)	mangrove, agricultural, urban areas	(Silva et al., 2019)
	Vibrio cholerae	USA (Chesapeake Bay)	estuarine	(Laverty et al., 2020)

	Vibrio mimicus Vibrio parahaemolyticus	Brazil (Guanabara Bay) North and Baltic sea	mangrove, agricultural, urban areas estuarine and coastal areas	(Silva et al., 2019) (Kirstein et al., 2016)
	Vibrio parahaemolyticus Vibrio vulnificus	USA (Chesapeake Bay) Brazil (Guanabara Bay)	estuarine mangrove, agricultural, urban areas	(Laverty et al., 2020) (Silva et al., 2019)
	Vibrio vulnificus	USA (Chesapeake Bay)	estuarine	(Laverty et al., 2020)
	Vibrio spp	Singapore	coastline	(Curren et al., 2019)
	Vibrio spp	UK (Scotland)	bathing beaches	(Rodrigues et al., 2019)
	Vibrio spp	China (Sungo Bay)	mariculture, shoreline	(Sun et al., 2020)
Epsilon Proteobacteria	Arcobacter spp	UK (Humber estuary)	estuarine	(Harrison et al., 2014)
	Arcobacter spp	USA (Chicago)	streams	(McCormick et al., 2016)
	Arcobacter spp	Singapore	coastline	(Curren et al., 2019)
	Arcobacter spp	Ireland (Carlow)	river	(Murphy et al., 2020)
	Arcobacter spp	China (Beijing)	sewage	(Wang et al., 2021)
	Helicobacter spp	Ireland (Carlow)	river	(Murphy et al., 2020)
Actinobacteria	Corynebacterium spp	China (Shanghai)	landfill leachate	(Su et al., 2021)
	Mycobacterium spp	China (Beijing)	sewage	(Zhao et al., 2020)
	Mycobacterium spp	China (Beijing)	sewage	(Wang et al., 2021)
	Mycobacterium spp	China (Henan province)	soil	(Yi et al., 2021)
Clostridia	Clostridium perfringens	Ireland (Carlow)	river	(Murphy et al., 2020)
	Clostridium spp	Singapore	coastline	(Curren et al., 2019)
	Clostridium spp	China (Guangzhou)	lake water	(Gong et al., 2019)
	Clostridium spp	China (Beijing)	sewage	(Wang et al., 2021)
Flavobacteria	Chryseobacterium spp	China (Guangzhou)	lake water	(Gong et al., 2019)

^{*} Environmental type refers to both *in situ* sampling and microplastic exposure to environmental samples under experimental conditions.

Figure captions

Figure 1.

Microplastics are pervasive pollutants found in virtually all environments (black diamonds: 1- aquatic systems, such as oceans, rivers, ponds or swamps; 2- terrestrial systems, such as agricultural land, meadows or forests). Sources of microplastic pollution are also widespread, involving plastic emission from (asterisks): 1- cities, villages, 2- roads, 3- sewage sludge, 4- atmospheric deposition. Particles smaller than 1μm can persist as suspended particles in the air. Environmental effects of microplastic pollution involve a diversity of mechanisms. (A) and (B) refer to the possible consequences of microplastic pollution on disease emergence in two specific environments, as detailed in Figure 2 and 3, respectively.

Figure 2.

Bacteria along with fungi or algae can readily form a biofilm on microplastics (here only bacteria are represented to not overload the figure). First, bacteria, both resistant (red cells) and susceptible ones (blue cells) are attaching to the fragment of microplastic (1). Binding of trace metals (2) and antibiotics (3) to microplastics can strengthen the selection on resistance genes, resulting in increased exposure of humans to multi-drug resistant bacteria. Biofilm i) confers protection from environmental stress and enhance bacterial persistence, ii) favors horizontal gene transfer potentially promoting the spread of resistant bacteria over susceptible ones.

The combination of enhanced bacterial persistence and the longevity of microplastics makes them ideal vehicles of potentially pathogenic bacteria from polluted sites (e.g., wastewater treatment plants where microplastics, antibiotics, trace metals, multi-drug

resistant bacteria can be found at high concentration) to natural environments. Figure 3. Microplastics can exert direct and indirect effects on invertebrate vectors (e.g., mosquitoes) or intermediate species (e.g., snails) of human infectious diseases (e.g., malaria, dengue, schistosomiasis, river blindness). Direct toxicity of microplastics on vector's life history traits might result in a reduction of disease transmission risk. However, the disruption of vector homeostasis due to plastic debris and/or the interference of the plastisphere with the vector's microbiota might also result in a better vectorial capacity, and increase the risk of disease transmission. Even in the absence of toxicity in vectors, microplastic pollution might affect their population dynamics through changes in the abundance of interacting species (i.e., predators and competitors), producing an increased risk of disease transmission.



Figure 1

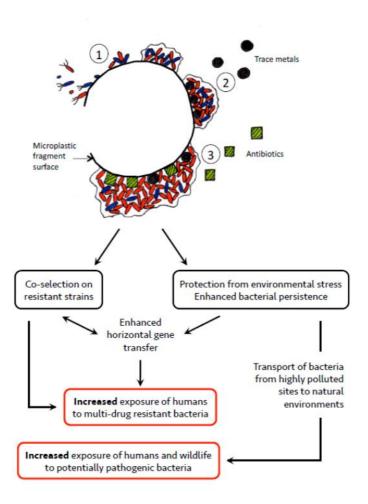


Figure 2

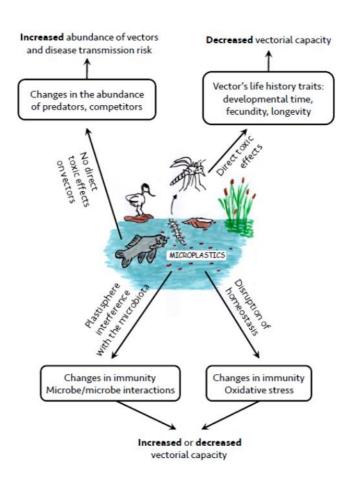


Figure 3